St. Bartholomew's Hospital Journal.

"Æquam memento rebus in arduis
Servare mentem."
—*Horace*. Book ii, Ode iii.

VOL. XLIII.—No. 10.] JULY 1ST, 1936. PRICE NINEPENCE.

CALENDAR.

Wed., July	1.	—Surgery: Clinical Lecture by Mr. Roberts. Tennis: Final of Inter-Hospitals Cup.
Fri., „	3.	—Dr. Hinds Howell and Mr. Harold Wilson on duty.
Sat., „	4.	—Cricket Match *v.* Richmond. Home. Tennis Match *v.* Staff College. Home.
Sun., „	5.	—Tennis Match *v.* Grasshoppers. Away.
Tues., „	7.	—Dr. Gow and Mr. Girling Ball on duty. Cricket Match *v.* Guy's Hospital. Away. Tennis Match *v.* Melbury Club. Away.
Fri., „	10.	—Dr. Graham and Mr. Roberts on duty.
Sat., „	11.	—Cricket Match *v.* Shoeburyness Garrison. Away. Tennis Match *v.* Guy's Hospital. Away.
Tues., „	14.	—Dr. Geoffrey Evans and Mr. Vick on duty.
Wed., „	15.	—Cricket Match *v.* St. Ann's. Away. Tennis Match *v.* R.N. College. Home.
Fri., „	17.	—Prof. Witts and Prof. Paterson Ross on duty.
Mon., „	20.	—**Last day for receiving matter for the August issue of the Journal.**
Tues., „	21.	—Dr. Hinds Howell and Mr. Harold Wilson on duty.
Thurs., „	23.	—Cricket Match *v.* Midhurst. Away.
Fri., „	24.	—Dr. Gow and Mr. Girling Ball on duty.
Sat., „	25.	—Tennis Match *v.* Melbury Club. Home.
Tues., „	28.	—Dr. Graham and Mr. Roberts on duty.
Wed., „	29.	—Cricket Match *v.* Hornsey. Away.
Fri., „	31.	—Dr. Geoffrey Evans and Mr. Vick on duty.

EDITORIAL.

THE General Council of Medical Education and Registration have adopted a report of the Curriculum Committee which will involve drastic changes in medical education, the most important of which is the raising of the registration age from 17 to 18. This, of course, implies that no man will be qualified until the age of 23, but it is difficult to understand how, without lengthening the period of clinical instruction, it will materially ameliorate the present position.

It is claimed, of course, that the general education of the student will now continue for one year more than in the past, but the period of training for his ultimate practical use remains unchanged. He will still spend two years at anatomy and physiology, and three years in clinical studies—and there, to our way of thinking, lies the discrepancy which could so easily be adjusted by abbreviating the curricula of the so-called "preliminary" subjects, and reducing by one-third or even more the knowledge of anatomy and physiology required of the student, whose eventual aim is general practice. For the intending surgeon, the Primary Fellowship would remain as a first step to the Final, and for the physician the Membership Examination would continue to fulfil its present purpose.

The Committee recommend that throughout the second year the student shall receive instruction designed to effect the association between pre-clinical and clinical subjects, and this will include methods of clinical examination, including physical signs, the use of the stethoscope, ophthalmoscope, auriscope and similar instruments, and the examination of normal body fluids. This is a point upon which great emphasis was laid by Lord Horder in his lecture on "Clinical Medicine" published in our columns in January, and it would clear the field for clinical instruction in the wards.

It is recommended, furthermore, that during the three-year period of clinical studies students should reside for a part of the time in hospital, in addition to the time already given to instruction in midwifery—but this would surely be less necessary where a residential college for students is available within or very near the hospital precincts.

It remains, then, a matter of individual opinion whether a year more devoted to general education or clinical instruction would be the more valuable in equipping men to practise general medicine and surgery, and the question of modifying the relative amounts of time devoted to special subjects is one of adjusting medical instruction to the widespread advances in medical science.

We have received the following from Dr. Gow and commend it very strongly to our readers:

ROYAL MEDICAL BENEVOLENT FUND.

To the Editor, 'St. Bartholomew's Hospital Journal'.

SIR,—To mark the Centenary Year of the Royal Medical Benevolent Fund, a special appeal is being made this year for two purposes:

1. For new subscribers to raise the annual income of £14,000 to £20,000. This would enable allowance to medical practitioners, who are in financial difficulty due to sickness or infirmity, to be raised from £40 to £52 per annum, and to widows and families left without adequate provision from £26 to £36 per annum.

2. For special donations to create a fund from which grants can be devoted, at the discretion of the Committee, to—

(*a*) Very urgent and distressing cases.

(*b*) Towards training the widows and orphan sons and daughters of medical practitioners to enable them to be self-supporting.

So far, in spite of the splendid lead given by Sir Thomas Barlow, the result of the appeal has been disappointing, and I ask Bart.'s men to do their utmost to support this most deserving charity.

Donations may be sent to me; annual subscriptions may be paid by bankers' order or by cheque sent to me or direct to Hon. Treasurer, British Medical Benevolent Fund, 11, Chandos Street, Cavendish Square, W. 1.

I am,
Yours faithfully,
A. E. GOW,
Hon. Local Secretary.

3, Upper Harley Street,
London, N.W. 1;
June 5th, 1936.

* * *

We note with great pleasure among the Birthday Honours the posthumous award of the Albert Medal in Gold to John Melly, M.C., F.R.C.S., "in recognition of the conspicuous gallantry which he displayed during the disorders in Addis Ababa in May, 1936, in his efforts to rescue British and other foreign nationals and wounded Abyssinians, in the course of which he received injuries from which he has since died."

* * *

We should like to congratulate Viscount Wakefield, C.B.E., on the award of the G.C.V.O., and Henry Harold Scott, M.D., F.R.C.P., on the C.M.G.

* * *

We record with great regret the death of Dr. W. H. Hurtley. An obituary notice will appear in our next issue.

* * *

Mr. G. C. Knight has been reappointed a Leverhulme Scholar of the Royal College of Surgeons.

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As we go to press, the Hospital and medical world has lost a great figure by the tragic death of Mr. H. P. Nelson. An obituary notice will appear in our next issue.

GASTRIC SECRETION IN HEALTH AND DISEASE.

(*Concluded from p.* 171.)

ANÆMIA.

The only two diseases which are directly connected with the gastric secretion are pernicious anæmia, which occurs only in the absence of gastric secretion, more especially the pyloro-duodenal secretion, and peptic ulceration, which occurs only in the presence of gastric secretion. Recent research has shown that hydrochloric acid has nothing to do with pernicious anæmia, and I should not be surprised if the same proved to be true of peptic ulcer. The gastric lesion in pernicious anæmia appears to be incurable, and though the patient may be maintained in good health indefinitely by preparations of liver or stomach, treatment is essentially substitution therapy and not cure. On passing the gastroscope on patients with pernicious anæmia in remission the gastric mucosa is found to be paler and smoother than normal, the rugæ are less evident, and the network of submucosal vessels is visible through the atrophic mucous membrane. The picture is not pathognomonic of pernicious anæmia, being seen in most cases when no acid is secreted after histamine, and in pernicious anæmia we must believe that a similar process has involved the duodenum. In idiopathic hypochromic anæmia apparent achlorhydria is present in 80%, and true achlorhydria in only 50%. The gastric lesion is much less constant than in pernicious anæmia, and the gastroscope reveals every grade of lesion from an atrophic mucosa identical with pernicious anæmia to an active gastritis of a hypertrophic type. My experience of idiopathic hypochromic anæmia now extends over a number of years, and I have been impressed to learn how rarely patients with this disease develop pernicious anæmia, although the gastric lesion seems so similar.

We are so accustomed in medicine to assign a single cause to a disease that it is a little difficult to realize that a clinical entity like idiopathic hypochromic anæmia may have a complex ætiology. There are at least three distinct factors in idiopathic hypochromic anæmia, and the weight of each may vary in different cases. The first is a diet deficient in iron. The second is impaired absorption of iron, owing to achlorhydria or extreme hypochlorhydria. The third is excessive loss of iron in menstruation and pregnancy. There is no doubt that the last factor is far and away the most important, as is shown by the facts that the

complete syndrome of idiopathic hypochromic anæmia, with glossitis, koilonychia, splenomegaly and microcytosis, rarely if ever occurs in the male, and that in the female the disease spontaneously disappears after the menopause. We are therefore not justified in regarding idiopathic hypochromic anæmia as a disease of the stomach, but must regard it as a disease of the reproductive epoch.

GASTRITIS.

In the last few years, as a result of the opportunity of examining portions of the fresh stomach removed by the surgeon, the introduction of methods of preserving the stomach from degeneration after death and the perfection of the gastroscope, much interest has been taken in the subject of gastritis. Hence gastritis, which was at one time an unorthodox diagnosis, has become rather too fashionable. People seem to be in danger of forgetting that gastritis is usually a silent lesion, and that whatever the gastroscope or the microscope may reveal, the great mass of dyspepsia is due to bad habits and disturbed emotions, and is to be treated by considering the individual as a whole and not by concentrating on the gastric mucosa. I shall always remember a man who was admitted to Guy's Hospital after trying to commit suicide by drinking lysol. His acute symptoms subsided within a few days, but three weeks later he vomited up most of the lining of his stomach. In that intermediate fortnight he had had no pain whatsoever, and in fact he told me that it was one of the happiest periods of his life—which had certainly been an unhappy one. He ultimately developed great contraction of the stomach, from which I think he died about a year later. There is no justification for regarding patients with low acidity as suffering from anacid gastritis, or patients with hyperchlorhydria as suffering from pre-ulcerous gastritis, or for treating them by diet and lavage, unless there is strong collateral evidence, for I have already pointed out that these are normal variations in the gastric secretion.

With the skilled assistance of Mr. H. W. Rodgers we have been able to inspect a large number of stomachs through the gastroscope, and it is my impression that a visible gastritis is less frequent in this country than on the Continent—perhaps because of differences in diet. Dr. H. A. Magnus has at the same time been studying the histology of material obtained at operation and autopsy, and he has found that a pyloric gastritis, often undetectable by the gastroscope, is very common, but that inflammation of the body of the stomach is unusual. It is very desirable that we should correlate the macroscopic and microscopic appearance of the stomach with the symptoms and with the gastric secretion. Stomachs obtained at operation or seen through the gastroscope are not a fair sample of the population, and the ideal thing would be to secure that in as many as possible of the patients who come to autopsy the notes record the presence or absence of digestive symptoms and the results of fractional gastric analysis. I often think that if ward clerks realized how valuable their notes may be for investigations of this kind, they would make them more complete than they do now.

For a simple account of gastritis I refer you to Knud Faber's lectures on "Gastritis and its Consequences", which were delivered at Guy's Hospital in 1934 at the invitation of the University of London. He divides chronic gastritis into a diffuse pangastritis which leads to atrophy and achlorhydria, and a juxta-pyloric gastro-duodenitis, which tends to erosion and ulceration, and is more likely to be accompanied by increase than decrease in the gastric secretion. Thus the pyloro-duodenal area, which is so different in function from the body of the stomach, reacts separately in disease. We probably make too much of ingested irritants as a cause of gastritis, for the stomach was designed to withstand considerable external insult. Few men abuse their stomachs more shamefully than Alexis St. Martin, but a temperate way of living soon restored his mucous membrane to normal. The stomach is much more susceptible to influences from within the organism, and it is the hæmatogenous toxins of the exanthemata, of acute rheumatism and influenza, and possibly of focal sepsis, which sow the seeds of chronic gastritis. Although I have indicated my own belief that gastritis is usually a silent lesion, it is of immense clinical importance in view of its possible relationship to that common and fatal disease, carcinoma of the stomach. It is largely because it may be a pre-cancerous lesion that we are making such efforts to simplify the diagnosis of gastritis, and that we are following up large series of patients with the diffuse pan-gastritis of idiopathic hypochromic and pernicious anæmia.

PEPTIC ULCER.

I have already demonstrated that the results of test-meals in large series of cases of peptic ulcer so closely overlap the normal, that it is difficult to explain the disease as due to a variation in one of the known constituents of the gastric juice, unless we are to lay greater stress than seems justified on the continuous nature of gastric secretion in that disease. We are in very much the same dilemma as we were in regard to pernicious anæmia before Castle and Meulengracht so

changed our outlook, and I do not think we shall solve the problem of peptic ulcer without some new knowledge of the same type. Peptic ulcer occurs only in situations where relatively undiluted gastric secretion is present, and for all practical purposes true achlorhydria excludes the diagnosis of simple peptic ulcer. Peptic ulcers occur in the stomach and first part of the duodenum; rarely in the œsophagus and small intestine when heterotopic gastric fundus mucosa occurs in these situations; in the jejunum and the ileum when the gastric juice is diverted there by anastomotic operations. It is remarkable how short is what one might call the striking distance of the juice, inasmuch as duodenal and jejunal ulcers occur only within a few inches of the stomach. The power of the juice to produce ulcers is lost before the acidity can have been greatly reduced. Indeed, as I have already indicated, I am very doubtful whether the acid is the principle in the juice that is responsible for the formation of ulcers. It is true that ulcers occur only in the presence of gastric juice, and that the pain of gastro-duodenal ulceration is relieved dramatically and constantly by strong alkalis such as sodium bicarbonate and magnesium oxide. On the other hand, fractional test-meals show that the acidity of ulcer patients who have had a gastro-enterostomy is almost as high as that of the general population, and large gastric ulcers heal as a result of duodenal feeding or jejunostomy—procedures which leave the juice unneutralized. I believe that healing in all these procedures is the result of rest. X-rays and gastroscopy show that peristalsis diminishes in the pyloric antrum after gastro-enterostomy, and the musculature may become atrophic.

Ideas of this kind, plus the realization of the difficulties and dangers of neutralizing the gastric acidity, are probably responsible for the decline in favour of the treatment of peptic ulcer by massive doses of alkalis. Dr. W. Oakley has shown that we cannot safely give more than 20 gm. of sodium bicarbonate or equivalent alkali a day. Theoretically this should neutralize 9 gm. of hydrochloric acid or 3 litres of juice. Ten 5-oz. feeds of milk neutralize another 1·5 litres, so that altogether our alkaline diet should neutralize 4·5 litres of juice. How is it that we do not succeed in maintaining neutrality, even allowing a generous margin over the normal 3 litres of juice? One reason is that secretion persists during sleep, when it is impossible to neutralize it, except by continuous drip feeding through a nasal tube—a method which has been used in America, but which reduces the medical treatment of ulcer to an absurdity. In the waking hours milk and alkalis are quickly evacuated by the stomach, and three-quarters of an hour after a milk feed, free hydrochloric acid may reappear in high concentration. The dose of alkali required to neutralize a given volume of gastric juice is always larger than the calculated chemical equivalent, owing to the reversible nature of the reaction. Moreover, the stomach seems to try and counteract the neutralizing action of alkalis by secreting more gastric juice, so that in the end the acidity may be higher with alkalis than without.

Various efforts have been made to overcome these difficulties. Hurst gives milky feeds hourly and alkali midway between feeds, but Emery suggests that it is better to make the feeds from a dried milk with which alkali has been incorporated. Others have suggested the use of slowly-acting neutralizing or adsorbent agents such as kaolin, aluminium hydroxide and magnesium trisilicate. I do not think the treatment of gastro-duodenal ulceration can advance far along these lines, and at present on the Medical Unit we do not prescribe alkalis unless pain is present, and never after hæmorrhage or in pyloric stenosis. Rest to the mind and the body is an essential, but it is rarely possible to keep patients in bed until their ulcers are healed, and if there is no pain and no blood in the stools, I like to give a diet of sufficient caloric content for the patient to be up part of the day or to go to a convalescent home. It has been said that peptic ulcers occur most commonly in those who drive vehicles, those who drive others, and those who drive themselves, and drugs of the barbiturate group help to relax tension. Belladonna and atropine, especially in the homœopathic dosage commonly employed, are of no value in easing spasm or diminishing secretion. The aims in composing a diet are to give foods which are non-irritating, and to ensure that the stomach is not empty or the ulcer exposed to undiluted gastric juice and the pangs of hunger. Milk forms the basis of the diet, and in order that it should not be too rapidly evacuated it should not be citrated, and feeds should be thickened with gruel or *purées* or given semi-solid as custards, blancmanges and junkets. Salt is allowed to taste but not to excess. It is just as grave a reflection on treatment for a patient with ulcer to feel hungry as for a patient with fever to have a dry tongue and feel thirsty, and to prevent this the diet should be adequate, and 5-oz. feeds may be given hourly. Cod liver oil before meals three times a day exercises the inhibitory action of fat on gastric secretion, and supplies vitamins A and D; other oils are used if it is not tolerated. Orange and tomato juice supply vitamin C, and marmite (given in milk), vitamin B. If there is anæmia, 30 gr. of iron and ammonium citrate are given thrice daily. Foci of infection are removed, but as imperfect mastication is just as harmful as

septic absorption, every effort is made to preserve serviceable teeth.

Loss of Gastric Juice.

In severe cases of gastro-duodenal ulceration systemic changes may occur, which have an immense influence on the vitality and resistance of the patient, and therefore on the risks of operation. It is only severe cases which require operation, and apart from perforation, no case of ulcer should undergo operation without a preliminary course of observation and treatment in a medical ward. It may be much more helpful for a patient to go to a convalescent home before operation than afterwards, for a man needs to be made fit to stand a big gastro-intestinal operation, and unless perforation or gross obstruction has occurred, there is no urgency. A considerable proportion of cases of peptic ulcer in the hospital classes ultimately require operation. If operation is restricted to those cases in which it is really essential, major procedures are required. The only way to reduce the mortality of these procedures is for physician and surgeon to co-operate, and to regard operation as an incident, albeit an important incident, in a disease process of unpredictable duration. With such co-operation the mortality of gastric surgery would be reduced as dramatically as that of thyroid surgery.

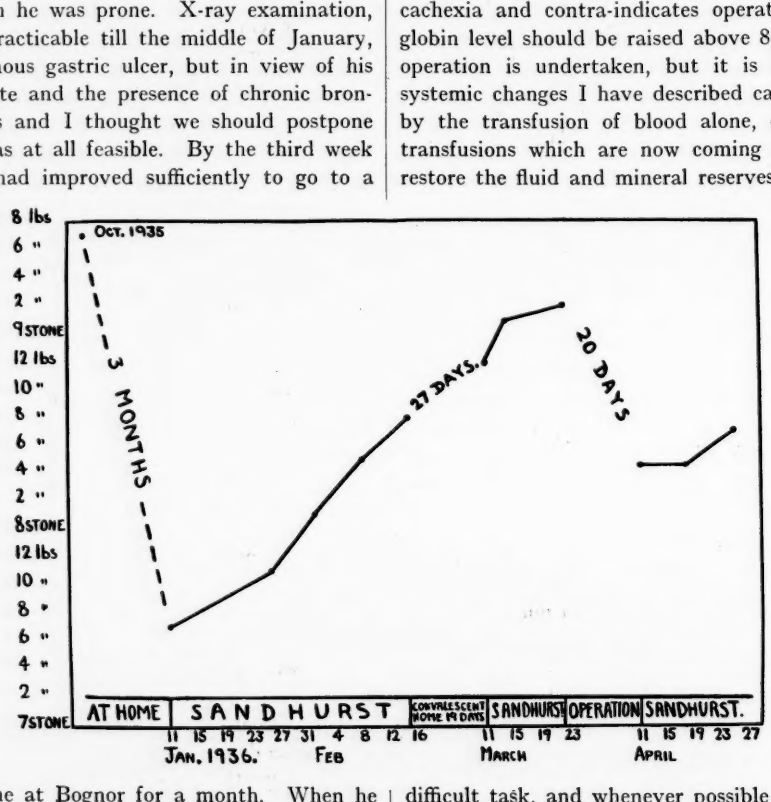
The bodily changes to which I have referred are brought about by malnutrition and avitaminosis; by the vomiting of gastric juice; by hæmorrhage; and by the therapeutic use of alkalis. Malnutrition and avitaminosis lead to wasting and loss of resistance to infection. I have already indicated that a single day's secretion of gastric juice constitutes a significant proportion of the total fluid and chlorine in the body. Repeated vomiting therefore leads to dehydration, to loss of mineral salts from the body, and to alkalosis owing to the unbalanced loss of acid. Hæmorrhage leads to anæmia and dehydration. Alkaline treatment leads to alkalosis. The main effects of these combined onslaughts on the body are wasting, dehydration, alkalosis and loss of mineral reserves, and for reasons not well understood these effects lead to uræmia and death. There is no short cut to their repair, and a patient who has become cachectic as a result of peptic ulceration needs many days of careful treatment before he is fit for operation. Mr. R. T. Payne, in a recent paper at the Paget Club, showed that the mortality of gastrectomy at this Hospital was high, and that it had not diminished in recent years. Deaths were not due to technical errors, but to pneumonia and lack of healing power in the tissues, and I think that these complications would be greatly diminished by more prolonged pre-operative treatment. The following case illustrates the lack of resistance in some of these patients:

A man of 52 who had had symptoms of duodenal ulcer since 1929 developed pyloric stenosis at the beginning of 1936. He had suffered from copious and projectile vomiting for two months before admission to the Medical Unit, and had been performing gastric lavage for one month. His general condition appeared satisfactory, blood-pressure 165/110, blood-count normal, blood urea 47 mgrm. per cent., alkali reserve 62 vols. carbon dioxide per cent., plasma chlorides 577 mgrm. per cent. While, however, his best weight was 10 st. 10 lb., his present weight was only 8 st. 2 lb. After further gastric lavage he was transferred to the Surgical Unit for gastro-enterostomy on the fifth day. The operation went very smoothly, but on the fourth day after the operation following an aperient his bowels were opened four times. On the fifth day his bowels were opened nine times, and his general condition deteriorated rapidly. The blood volume was diminished, as shown by the fact that the red cell-count had risen to 6·8 millions per c.mm. The blood urea was 320 mgrm. per cent. Next morning the patient rather suddenly collapsed and became dyspnœic, his extremities were blue and cold, his face shrunken, and his systolic blood-pressure was below 60 mm. Hg. The blood urea had risen to 440 mgrm. per cent.; the plasma chlorides had fallen to 483 mgrm. and the alkali reserve to 28 volumes per cent. Efforts to restore the circulation were of no avail and the patient died a few hours later. There was no autopsy.

Although this man's blood-count and blood chemistry were normal, his loss of approximately 25 per cent. of his weight was probably associated with such a great diminution in the fluid and mineral reserves of the body that he was unable to cope with a moderate diarrhœa, and quickly succumbed to uræmia and acidosis. In the following case perseverance with pre-operative treatment in the face of considerable difficulties was probably responsible for the success of the operation when it was finally performed:

Geo. F—, æt. 54, had been under treatment for a gastric ulcer for three years. As the ulcer grew larger on out-patient treatment, he was admitted to Rahere Ward in September, 1935, and had a month's treatment with careful diet and histidine. The ulcer crater appeared smaller at the end of this time, but he soon broke down on returning home. In November his pain became very severe, changing to attacks of severe griping colic that doubled him up. He had much vomiting, and finally, at the beginning of December, hæmatemesis and melæna which brought him into Sandhurst Ward. His pain was severe enough to suggest a leaking perforation, but there were so signs

of this. He was in miserable physical condition, the hæmoglobin was only 40 per cent., blood urea 80 mgrm. per cent., alkali reserve 76 vols. per cent. Following our current practice we gave a fairly full fluid diet right away, relieving his pain at first with morphia and nembutal. Later it was possible to give alkalis in sufficient dosage to relieve the pain, but avoid the alkalosis to which he was prone. X-ray examination, which was not practicable till the middle of January, showed an enormous gastric ulcer, but in view of his poor physical state and the presence of chronic bronchitis, Prof. Ross and I thought we should postpone operation if it was at all feasible. By the third week of February he had improved sufficiently to go to a convalescent home at Bognor for a month. When he came back in March the change in his general condition and the diminution in the crater of the ulcer were equally remarkable. Under local anæsthesia Prof. Ross successfully performed a partial gastrectomy—the ulcer was now about 1 cm. in diameter—and apart from a little bronchitis after the operation convalescence was uninterrupted. In the figure I have charted his fluctuations in weight during this period, to demonstrate that both relapse of the ulcer and operation are accompanied by loss of weight, and to suggest that operation should not be undertaken until the patient is on the upgrade.



In the assessment of the patient's condition the general appearance is of considerable value, but more precise data are provided by the hæmoglobin content of the blood, the blood chemistry and the loss of weight. Of the chemical changes in the blood the most constant is an increase in the blood urea; sodium and chlorine may be diminished and the alkali reserve increased. I attach great importance to loss of weight, as this is the best indication of loss of fluid, minerals and nutritional reserves. It is gauged by comparing the patient's best weight with his present weight. A loss of 25 per cent. in the body-weight usually indicates a considerable cachexia and contra-indicates operation. The hæmoglobin level should be raised above 85 per cent. before operation is undertaken, but it is obvious that the systemic changes I have described cannot be remedied by the transfusion of blood alone, even the massive transfusions which are now coming into fashion. To restore the fluid and mineral reserves of the body is a difficult task, and whenever possible it should be done by a course of dietetic treatment, whereby the organism can select the materials it requires. Infusions of saline may help, but are rarely sufficient in themselves to repair in a short time the complicated humoral changes. Alkalosis may be corrected by the oral administration of acid sodium phosphate, 30 gr. four-hourly. Ammonium chloride should not be used, as it increases the dehydration, and in any event the blood must be tested at frequent intervals to avoid overdosage. Of course patients should not be allowed to get as ill as in the following case, which illustrates the difficulties in trying to restore the internal milieu to normal:

A man of 60 had had dyspepsia suggestive of duodenal ulcer since 1932. X-ray examination in 1933 had not revealed an ulcer, but he was treated by diet and an alkaline powder, which was prescribed in a dosage of 60 gr. *t.d.s.* Three weeks before admission he began

to suffer from attacks of giddiness, and ten days later nausea, hiccough, vomiting and constipation set in. He ceased to take alkalis, but his condition deteriorated, his output of urine diminished and he grew drowsy and stuporose. On admission to Sandhurst Ward in January, 1935, he was obviously uræmic and the blood urea was 200 mgrm. per cent. It was decided that this was a gastric and not a renal uræmia, as the urine contained only an inconstant trace of albumen and no casts, the blood-pressure was only 150/82, and the alkali reserve was 90 vols. carbon dioxide per cent. He was treated with fluids, acid sodium phosphate, and later ammonium chloride, the urine volume increased to a satisfactory level, and his mentality cleared. Unfortunately we pushed the ammonium chloride too far, so that he developed acidosis and relapsed into coma and died. The last examination of the alkali reserve showed 40 vols. per cent., but as we did not hear the result till some hours later we probably depressed it much lower than this. Efforts to change the alkali reserve in dehydrated patients are fraught with danger and need very careful control. Autopsy showed an ulcer which had narrowed the duodenum so much that it admitted only the tip of the little finger, and chronic focal nephritis.

In another year I hope to discuss our observations on hæmatemesis and melæna, in which humoral changes are present in an aggravated form. Enough has been said to prove that in peptic ulceration we must not limit our gaze too closely to the stomach and duodenum, for the loss or neutralization of the gastric secretion produces widespread changes in the organism, and these secondary phenomena may be a greater menace to life than the actual ulcer.

L. J. WITTS.

ADVICE TO A DRESSER.

"Ye have read, ye have heard, ye have thought," he said,
"and the tale is yet to run:
"By the worth of the body that once we had, give answer—
what ha' ye done?"—KIPLING.

GOOD advice is like a bottle of medicine; it may never be taken, but if it should be it will probably do no harm, and it may occasionally do good. These remarks are addressed to every dresser in the knowledge that they may be read by a few and will be of real value to still fewer; but when these conditions are understood and accepted by both parties, no blame can be imputed either to the writer or to the majority of his audience.

YOUR FIRST APPOINTMENT.

Surgery is a craft, and the way to learn it is through apprenticeship to a master, by watching him at the bedside and in the operation theatre and then going away and trying to do likewise. From him you must learn how to take a history and how to elicit physical signs, but remember that it is only by practising these methods over and over again that you will train your own powers of observation. This is what one of our most renowned clinical teachers meant when he said to some *habitués* of the back row, "Feel it, gentlemen, feel it; you cannot learn by smell alone!"

Errors of diagnosis are attributable, not to ignorance, but to imperfect clinical examination. By the time a man becomes qualified there can be few diseases of which he has never heard, and if he makes sure of his physical signs he is almost certain to be able to interpret them correctly. He makes mistakes either because he has got the signs wrong, or more commonly because he has never thought of looking for them. The thorough examination of any part of the body must be orderly so that you may know when you have completed it, and you should try from the very beginning to be methodical in your clinical work. Your efforts in this direction will be greatly aided by the Little Grey Book.

Next you must learn to write a concise description of what you have observed. When Bacon wrote, "Reading maketh a full man; conference a ready man; and writing an exact man", he laid stress upon the importance of writing as an aid to memory. But note-taking has the added virtue that it makes you more accurate in your clinical examinations, for it is only when you try to write down the details of a history or of certain physical signs that you realize how many features can be missed at the bedside. You should try to make your notes brief yet comprehensive, using simple expressions and avoiding terms which imply a diagnosis and make you jump to an unwarranted conclusion. Always bear in mind the value of recording the absence as well as the presence of relevant symptoms and important signs.

You are certain to find much of interest in the operation theatre, but the cases which are commonly considered the most exciting are not necessarily the most instructive. You cannot see too many of the lesser operations of surgery, for you may be expected to perform them yourself in general practice, and the same applies to emergency operations. The profit to be derived from watching an artist or a craftsman at work depends upon knowing what to look for, and, in watching a surgeon, you should observe how closely his methods conform to the fundamental principles of

good technique—the maintenance of asepsis, gentleness in handling the tissues, and the effective control of hæmorrhage.

From what has already been said you will have gathered that you will learn more in your early days from your patients than from your books, for the best test of a man's knowledge of surgery will always be what he has done or seen, rather than what he has heard or thought or read. But pathology is a subject you must add to anatomy and physiology in order to understand the science of surgery, and I would advise you to read *Surgical Pathology* by Bowlby and Andrewes and *Tumours* by Bland-Sutton. Men may tell you that these books are out of date, but heed them not. They maintain their perennial freshness, and are a perpetual delight to the reader because they are models of pure English; and whereas the former is of special value to Bart.'s men because it is written about the specimens in our own Museum, the latter is important because it shows how much can be learnt about disease in man by considering the fowls of the air and the beasts of the field.

The Out-Patient Department.

As a ward dresser you have the opportunity of making a complete study of a number of cases, but in the Out-Patient Department you can make a less complete study of a much larger number. The examination of your patients is incomplete in that you are obliged to concentrate your attention particularly upon the local diseases of which they complain, and the time at your disposal is insufficient for detailed inquiry into all the systems of the body, and for the pathological investigations which are carried out in the wards. But the examination of the local lesion must be performed with the utmost care, and the practice and experience gained by the examination of many patients should so improve your clinical faculties as to ensure that accuracy shall not be sacrificed to speed.

Whereas in the earlier period of your training you should resist the temptation to make a diagnosis until you can arrive at your conclusion by a deliberate process of deduction from the observed facts, you should now train yourself to diagnose at sight. This method is of course more risky, and its value depends on the subsequent inquiry to prove or disprove the correctness of your first impressions; but provided you take the trouble to discover your mistakes, you will learn more by making them than by reserving judgment in order to avoid them.

It is a mistake to neglect cases in the Out-Patient Department which at first sight appear trivial. You must remember that in practice the majority of your patients will suffer from minor maladies which seldom reach the wards of a teaching hospital, and it is at this stage of your career that you must make yourself familiar with the manifestations and the treatment of these common complaints.

Your cases in the Out-Patient Department should also stimulate you to read one of the standard systems of surgery. The benefit reaped from reading a surgery book right through is not commensurate with the labour entailed, but to refer to a good text-book for further information about cases you have seen will clarify obscurities and amplify your knowledge, and will help to fix important facts in your memory. You will probably find that writing down a short *résumé* of what you have been taught and what you have read will help you to co-ordinate and remember the information you have collected from various sources.

Your Last Appointment.

It is a popular fallacy that a man who is doing his "second-time" dressership just prior to the final examination in surgery should not take cases in the wards. Some consider it beneath their dignity to write notes and do dressings, while others regard it as a waste of time. Wisdom and humility always go hand in hand, and the truth is that when you are nearly qualified you can learn more than ever before by studying your patients, though from a rather different point of view. In your earlier days you must necessarily concentrate your attention upon methods of examination, for accurate diagnosis must precede rational treatment. But you must remember that although to you it is a matter of the first importance to give the patient's disease a name, he is much more interested in what you are going to do for him and whether he is going to lose his job as the result of it, while his relations and friends will want to know whether the disease is likely to shorten his life. It is during the latter part of your out-patient work and while you are doing this final clinical appointment that you must devote special attention to these details of therapeutics and prognosis, and when you have finished your examination of a patient you must make a habit of asking yourself what you are going to do for him.

Study therefore your patients' temperature charts and their blue-boards, and attend the Follow-up Department. You must become familiar with the odd things a chart may show after an operation even when there is no cause for alarm, and you must learn to recognize its danger-signals and know how to search the patient for the source of the trouble. The blue-board will stimulate your interest in your patient's food—a matter about which you will be expected to

give detailed and authoritative advice in the first house you enter as the doctor; and from it you must also learn the few drugs which are necessary to prepare patients for operation and to alleviate their suffering afterwards. In the Follow-up Department you will learn the limitations of modern surgery—a matter which you can discover in no other way, and which you must know if you are to advise your future patients whether to submit to operation or to " dree their weird ".

Yet I know that even if I could speak with all the tongues of men and angels there would still be many unconvinced of the folly of reading in the library while the wards and out-patient departments are open. There is plenty of time for books when the wards are closed, but even then the Museum is a more profitable place to work in. You should persist in the habit of reading up your cases in text-books of surgery and pathology, but I want you to realize how valuable the *Museum Catalogue* may be in linking up clinical pictures with pathological processes, thus clarifying your ideas of disease as a whole.

Let me assure you that, in spite of what you might be led to assume from its popularity, axillary absorption is a very slow process, and a man must walk about with a book under his arm for many weary months before he can convince even the examiners that he knows the requisite minimum about clinical surgery. Kipling taught us a lesson which you would do well to lay to heart when he told how " the God that he took from a printed book " failed to get Tomlinson into either Heaven or Hell. So shall reading without clinical experience fail to help you either in your examinations, or in the more serious problems which await you in practice. After your studentship is over you will come to rely not on what you have heard or thought, but upon what you have seen with your own eyes and done with your own hands, and this experience can be gained only from continual contact with sick men and women.

J. PATERSON ROSS.

PITFALLS IN THE DIAGNOSIS OF TOXIC GOITRE.

THE incomplete forms of toxic goitre rival almost any disease in difficulty of diagnosis; their signs and symptoms may exhibit a striking similarity to those of other clinical entities. This may often not be recognized; consequently partial thyroidectomy may be recommended in cases in which, on closer observation, it is very doubtful whether the diagnosis of toxic goitre is correct. The commonest condition to be confused with mild toxic goitre is the so-called neuro-circulatory asthenia, or cardio-vascular neurosis, the underlying pathology of which is considered to be a derangement of the function of the autonomic nervous system—the " autonomic imbalance" of Kessel and Hyman (1). In fact these authors have gone so far as to say that the only difference between toxic goitre and " autonomic imbalance " is the absence of a raised basal metabolic rate in the latter. It is proposed in this paper to discuss various points which may be of assistance in differentiating between the two diseases, but it must be admitted that in some cases this is not always possible. Reference will also be made to several other conditions which may occasionally be mistaken for toxic goitre, and to one or two conditions which may be mimicked by atypical forms of toxic goitre.

Neuro-circulatory asthenia and neurasthenia.—The writer has recently studied the symptomatology of all those cases admitted to the wards of the Medical Unit during the last five years, amounting to 25 in number, which presented features almost indistinguishable from those of mild toxic goitre, and which were diagnosed as neuro-circulatory asthenia, cardio-vascular neurosis or neurasthenia. At the same time analysis was made of the signs and symptoms of an equal number of the cases which presented similar features to the former group and in which the diagnosis of toxic goitre was made.

The signs and symptoms which occurred in both groups consisted of nervousness, a change in disposition, periods of faintness and giddiness, headaches, sweating, hot flushes, a feeling of warmth, dyspnœa on exertion, palpitations, præcordial pain, tiredness, irregular or scanty menstrual periods or amenorrhœa, loss of weight, enlargement of the thyroid, tremor and tachycardia. The symptoms which were commonest in the two groups, occurring in more than half of the cases, were nervousness, dyspnœa, palpitations, tiredness, enlargement of the thyroid and tachycardia, whereas præcordial pain and giddiness were rare occurrences. Changes in disposition, faintness, sweating, hot flushes and a feeling of warmth occurred about equally in the two groups. Headache was far more frequent in neuro-circulatory asthenia and neurasthenia than in toxic goitre, being present in 17 of the 25 cases, compared with 5 of the 25 cases of toxic goitre. It was frontal, vertical, occipital or temporal and was usually throbbing in character. Menstrual disturbances occurred in about half of the cases of toxic goitre and in only one-quarter of the cases of neuro-circulatory asthenia. The more important signs and symptoms which may or may not

have a bearing on diagnosis will be briefly discussed individually.

Lost of weight.—In the early cases of toxic goitre this symptom may not be present, but in the analysis of the cases under discussion it was found to be far more common in toxic goitre than in neuro-circulatory asthenia or neurasthenia. It occurred in 15 of the patients with toxic goitre and in only 6 of those diagnosed as neuro-circulatory asthenia or neurasthenia. The loss of weight in toxic goitre occurred in spite of the appetite being good, whereas in neuro-circulatory asthenia this symptom was frequently associated with a poor appetite and may have been partly due to this cause.

Exophthalmos.—The presence of exophthalmos is strong evidence in favour of the diagnosis of toxic goitre, but it must be borne in mind that some patients may have normally prominent eyes and inquiry should be made concerning this. Such prominence of the eyes occurred in two of the cases diagnosed as neuro-circulatory asthenia. However, in mild toxic goitre exophthalmos is often absent; it was evident in only 11 of the 25 cases diagnosed as such. Careful observation may discover a slight stare or a glistening of the sclera which is unusual in normal persons, and the patient should be asked whether her friends have remarked upon such a condition. The stare in mild cases of toxic goitre is important and should be carefully considered. If it be established, it may be a deciding factor in favour of the diagnosis of toxic goitre. It was present in 8 of the cases of toxic goitre in this series.

Goitre.—Simple enlargement of the thyroid gland may occur in a patient with neuro-circulatory asthenia or neurasthenia as in any other person. It cannot be too strongly emphasized that the presence of an enlarged thyroid does not necessarily clinch the diagnosis of toxic goitre in patients who present symptoms suggestive of toxicity. In the present series enlargment of the thyroid gland was evident in 21 cases of toxic goitre, and in 16 of those diagnosed as cardio-vascular neurosis, neuro-circulatory asthenia or neurasthenia. If a history can be obtained that the thyroid enlarged at about the same time as the other symptoms appeared, it is suggestive that these may be due to toxic goitre. On the other hand, patients may have thyroid intoxication with no enlargement of the thyroid, a possible explanation of this being that a high iodine intake with the food and easy absorption from the gut enables the thyroid to secrete increased amounts of thyroid hormone without the necessity of enlarging in the process. If thyrotropic hormone of the anterior pituitary be injected into guinea-pigs the thyroid will become enlarged and hyperplastic, and the basal metabolic rate will rise in consequence of increased secretion of thyroid hormone. If, however, iodine be given at the same time as thyrotropic hormone, hyperplasia and enlargement of the thyroid will not take place, but the basal metabolic rate will still rise, in some cases even higher with than without iodine.

Tremor.—Tremor of the fingers of the outstretched hands may be present in both diseases, but it was twice as frequent in toxic goitre (14 cases) as in neuro-circulatory asthenia (7 cases). In toxic goitre the tremor is characteristically fine, whereas in neuro-circulatory asthenia it is usually coarse.

Tachycardia.—Little can be said concerning this sign in diagnosis. With the patient at rest the pulse-rate during sleep on the whole approaches the normal level in neuro-circulatory asthenia, whereas in toxic goitre it continues to be raised; this, however, may not always be the case. In both conditions the pulse-rate falls to a lower level after rest in bed and as the patient becomes used to ward conditions. The character of the pulse in toxic goitre is typically bounding and of good volume, whereas in neuro-circulatory asthenia the volume may be poor. The cardiac impulse was described as forcible or overacting in 14 cases of toxic goitre, but in only 3 cases of neuro-circulatory asthenia.

Previous history.—With regard to the previous history of these patients, both conditions may arise following a period of mental stress, anxiety or overwork. Perhaps the patient with neuro-circulatory asthenia is more likely to have been "always delicate" or to have had "nervous breakdowns" in the past than the patient with toxic goitre, but as a similar history may be obtained in toxic goitre, it is of little help in diagnosis.

An analysis of the two groups of patients leads to the conclusion that in the differentiation of cases which present the difficulties which have been described, loss of weight accompanied by a good appetite, a slight stare or glistening of the sclera, a tachycardia with a high pulse-rate during sleep and a forcible cardiac impulse, a fine tremor and the simultaneous appearance of thyroid enlargement with the other symptoms are points in favour of the diagnosis of toxic goitre. Absence of exophthalmos and of thyroid enlargement does not contra-indicate such a diagnosis, and the presence of thyroid enlargement does not necessarily support it.

Basal metabolic rate.—The basal metabolic rate is not always an index of the degree of thyrotoxicity, and for this reason no reference has yet been made to its value in the diagnosis of the conditions under discussion. The basal metabolic rate in normal persons varies from −10 to +10%—a normal "highly strung" person may have a basal rate as high as +15 or +20%. The occurrence of toxic goitre in a person with a normally high metabolism will cause an increase in basal

rate to a figure above the normal limit, but a person with a normally low basal rate and with an equal degree of thyrotoxicity will show a figure within normal limits. For this reason the basal metabolic rate may be of little value in differentiating toxic goitre from neuro-circulatory asthenia, for the readings in both conditions may be at a high normal level. Further, a patient with neuro-circulatory asthenia may be so nervous when the test is performed that a figure considerably above the normal limit is obtained. A considerable amount of discretion, therefore, must be used in interpreting the basal metabolic rate. A low figure, *i. e.* one below or just above zero, is in favour of the case not being one of toxic goitre.

It is probable that toxic goitre and neuro-circulatory asthenia are closely related, and in borderline cases it may be impossible to come to a definite decision; in such cases conservative measures of treatment should always be undertaken and the patient kept under observation. A few months later the evidence in favour of a diagnosis of toxic goitre may be less indefinite.

The following summaries of case-reports briefly illustrate some of the points under discussion:

(1) Female, æt. 21. Family nervous and "highly strung"; at puberty patient had a goitre which disappeared when she was aged 18. About two years before admission she became miserable and nervous, with palpitations, dyspnœa on exertion, frontal headaches, occasional præcordial pain, irregular menstrual periods, some loss of weight, anorexia and dysphagia. On examination, no exophthalmos; slight tremor of fingers; thyroid moderately enlarged; pulse-rate 100–110, settling to 80–90 on rest in bed; basal metabolic rate + 53% (accuracy of reading doubtful). Diagnosis: Neurasthenia.

(2) Female, æt. 35. In the past had occasional "breakdowns"; work monotonous. Six months' history of nervousness, fainting attacks, palpitations, sweating, hot flushes, a feeling of warmth, menses slightly irregular; no loss of weight; no exophthalmos; coarse tremor; no thyroid enlargement; pulse-rate 90–100; basal metabolic rate + 3%. Diagnosis: Neurasthenia. The diagnosis in this case depended on the basal metabolic rate.

(3) Female, æt. 29. Never robust; in the past many "nervous breakdowns"; much quarrelling at home. For the last 15 years patient has been excitable, emotional, introspective; palpitations; headaches; no loss of weight; menses regular; ? slight exophthalmos; no tremor; very slight thyroid enlargement; pulse-rate 80–100; basal metabolic rate + 14%. Diagnosis; Neurasthenia. A border-line case.

(4) Female, æt. 24. One year's history of fainting attacks, which were her chief complaint; attacks began with stabbing pain over the præcordium, followed by numbness of the legs, a tight sensation over the face, giddiness and unconsciousness, which might last for a quarter to two hours; patient had four such attacks. Dyspnœa on exertion; occasional præcordial pain while sitting down; palpitations at night; appetite varied, epigastric fullness after food, flatulence and heart-burn; twitching of arms and legs while in bed; sweating and hot flushes; slight loss of weight. On examination no exophthalmos; no tremors; skin warm and moist; pulse-rate 80; thyroid slightly enlarged. The diagnosis of neuro-circulatory asthenia was made. Six months later patient began to tire easily; sweating marked; marked tachycardia on slight exertion; slight stare, but no definite exophthalmos; slight tremor, thyroid slightly enlarged; pulse-rate 100; basal metabolic rate not estimated. Diagnosis: Toxic goitre.

(5) Female, æt. 52. Six months' history of nervousness, dyspnœa on exertion, palpitations, occasional headaches, some loss of weight; appetite good; menopause four years previously; no exophthalmos; slight tremor; slight thyroid enlargement; pulse-rate 80–120, during sleep 80–100; basal metabolic rate + 58%. Diagnosis: Toxic goitre.

(6) Female, æt. 51. Always "delicate"; six years' history of depression, nervousness, dyspnœa on exertion, palpitations, tiredness, loss of weight; appetite good; frontal headaches; præcordial pain occasionally; no exophthalmos; fine tremor; thyroid enlarged; pulse-rate 90–100, during sleep 90; cardiac impulse forcible; basal metabolic rate + 38%. Diagnosis: Toxic goitre.

(7) Male, æt. 31. One year previously had "influenza" followed by weakness, tiredness and lack of energy; no dyspnœa; occasional palpitations; no sweating, no hot flushes; appetite good, but during the last four months lost 2 st. in weight; no nervousness; exophthalmos; skin warm and moist; no tremor; thyroid diffusely enlarged; pulse-rate 80–120, during sleep 80–90; basal metabolic rate + 21%. Diagnosis: Toxic goitre.

Of the various conditions which may be confused with toxic goitre, the menopause, arterio-sclerosis and pulmonary tuberculosis should be mentioned, and in a few cases toxic goitre may bear a close resemblance to Addison's disease.

Menopause.—A woman, æt. 46, had a ten months' history of a tight feeling in the neck, tiredness and palpitations; no nervousness, no sweating, no hot flushes, but a feeling of warmth; frequent frontal and vertical headaches; the menses were becoming irregular; no loss of weight. On examination, no exophthalmos;

pulsation at the root of the neck, but no thyroid enlargement; skin warm and moist; fine tremor of the fingers; pulse-rate 90–120, during sleep 90. At first the diagnosis in this case was toxic goitre, but later when the basal metabolic rate was found to be −2 and −1% on two occasions it was considered that her signs and symptoms were manifestations of the menopause.

Arterio-sclerosis.—The symptoms of the following case, at first thought to be due to toxic goitre, were ultimately considered to be the result of arteriosclerosis. It is well known that arteriosclerosis and hypertension may produce a clinical picture similar to neurasthenia, and on this account, especially if associated with thyroid enlargement, may be mistaken for toxic goitre.

The patient was a female, æt. 51, with one year's history of palpitations, dyspnœa on exertion, hot flushes, occasional headaches, dyspepsia, no nervousness, no loss of weight. On examination: Anxious type; no exophthalmos; slight enlargement of the thyroid; pulse-rate 80–90; cardiac impulse forcible; blood-pressure 200 systolic, 112 diastolic; basal metabolic rate in a surgical ward + 31%, but later after the patient was transferred to a medical ward − 15%.

Pulmonary tuberculosis.—Patients in whom the predominant and sometimes only feature is tachycardia frequently cause some difficulty in diagnosis; in such cases pulmonary tuberculosis should always be seriously considered. The writer was asked to see a female patient who was thought to be suffering from mild toxic goitre. Æt. 25, she had one year's history of palpitations, sweating, occasional slight cough, but no sputum, loss of weight, appetite moderate, menses irregular; she was always of a nervous disposition. On examination: No exophthalmos, coarse tremor, thyroid slightly enlarged; pulse-rate 90; there were doubtful signs at the right apex, but an X-ray of the chest showed a tuberculous lesion in this position.

Addison's disease.—In addition to the incomplete forms of toxic goitre there are several peculiar types which may give rise to difficulty. One of these is the so-called adrenal type, termed by some writers "apathetic hyperthyroidism" (2), the clinical features of which may be highly suggestive of Addison's disease. That a brownish-yellow pigmentation sometimes occurs in toxic goitre is well known, and frequently in toxic goitre asthenia is a marked feature. It is when pigmentation and asthenia occur together, in addition to mental apathy instead of the more usual restlessness and nervousness, that confusion is likely to arise. In this type, which occurs in patients of middle age or past middle age, there may be no exophthalmos and little staring of the eyes, the thyroid may be small, the pulse-rate is usually not higher than 100–120 and the cardiac impulse is not forcible. The skin is relatively dry and cool, unlike the warm moist skin of the more usual variety, and tends to be pigmented. There is a considerable degree of asthenia, and the mental condition appears to be one of repose or apathy. Sometimes, however, the information may be obtained that although the patient is seemingly reposeful her emotions are "pent up". In elucidation of such a case a raised basal metabolic rate is in favour of toxic goitre.

Therapeutic test of iodine.—In cases of difficulty the therapeutic test of the administration of iodine may be of assistance (3). The patient should be confined to bed, and after a preliminary rest period of about two weeks should be given Lugol's solution, ℥v, three times daily. If the patient has not a nodular goitre and if she has not received iodine recently, there will be a considerable fall in pulse-rate after iodine administration if she is suffering from toxic goitre, but not if she is suffering from some other disease.

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A. W. SPENCE.

PERFORATED PEPTIC ULCERATION.

THE perforation of a peptic ulcer into the general peritoneal cavity is an event which, unless recognized early and dealt with promptly by surgical means, inevitably leads to death. After perforation the patient may live for 72 hours or longer, and during this period presents a constantly changing clinical picture. The fact that the clinical picture is dynamic rather than static is at times overlooked, and this alteration corresponds to the peritoneal and systemic pathological changes which follow the abdominal catastrophe. In general the abdominal catastrophe is so dramatic and so acute that the doctor is likely to be called to the patient shortly after its occurrence. In these circumstances recognition of the true state of affairs should not give rise to serious difficulty. On the other hand, the patient may come under observation during any of the subsequent phases between perforation and death, and unless the changing clinical picture is remembered it is still possible for diagnostic errors to occur.

This transition from normal health to the moribund stage associated with a Hippocratic facies is a gradual one, but it is possible to divide this downward progress into various phases. These inevitably tend to overlap, but for purposes of description may be considered as follows: the actual moment of perforation, and the stages of chemical peritonitis, reaction, bacterial peritonitis, and, finally, paralytic intestinal obstruction.

The Moment of Perforation.

Pathologically, the perforation of a peptic ulcer usually represents the perforation of a chronic ulcer as the result of an acute exacerbation of the ulcerative process. Occasionally it occurs as a complication of acute ulceration, but this is very rare.

Clinically, the moment of perforation gives rise to sudden, intense upper abdominal pain which rapidly becomes generalized, and on account of its severity leads to collapse. The patient usually falls to the ground and is unable to move to assist himself. Few acute abdominal conditions are so dramatic and so sudden in their onset as perforation of a peptic ulcer —an event which may in the space of a few seconds bring an apparently healthy individual to helplessness and anguish. Acute pancreatitis is the only other acute abdominal condition which may at times give rise to a closely similar clinical picture.

The physical signs following perforation will be considered under the next section dealing with chemical peritonitis.

The Stage of Chemical Peritonitis.

As the result of perforation of the stomach or duodenum, gastric or duodenal contents are poured into the peritoneal cavity. The insult to the peritoneum produced in this way is primarily a chemical one, and arises from the presence of hydrochloric acid and other digestive juices. At this stage the peritonitis is a chemical irritation rather than a bacterial phenomenon.

The stage of chemical peritonitis generally lasts for about six hours before passing gradually into the stage of reaction. During this time the patient suffers intense generalized abdominal pain. Nausea and vomiting are both exceptional. The patient lies motionless on the couch, usually with his legs drawn up, and is terrified to carry out any movement. The face is pale, the lips are blanched and the skin generally is cold and clammy. The temperature during this stage is subnormal, and may often be as low as 96°. On the other hand, despite the profound collapse and the almost shock-like condition which is present, the pulse shows surprisingly little alteration. A slight increase usually occurs, but the rate rarely exceeds 90 per minute, and is often less than this. The respiratory rate is almost always increased owing to the absence of abdominal and diaphragmatic movements. Though the clinical condition bears some resemblance to true surgical shock, there is a fundamental difference in the fact that the pulse-rate is only slightly increased. The term "peritoneal shock" may be used to describe the clinical condition.

On examination of the abdomen at this stage retraction may be present, and this is especially likely to be the case with perforation of a gastric ulcer where a good deal of wasting has taken place. No abdominal movement occurs with respiration. On palpation there is generalized rigidity, which is invariably board-like. Anything more than the lightest pressure produces exquisite pain. On percussion two things may be made out. The first is the diminution or even the complete absence of the liver-dullness owing to the presence of gas in the peritoneal cavity, and the second is the presence of dullness in the flanks or hypogastrium on account of the collection of fluid at these sites. No attempt should be made to elicit shifting dullness in this or any other acute peritoneal condition on account of the painfulness of this manœuvre, its uncertainty, and the risks of spreading infection to other parts of the peritoneum.

The Stage of Reaction.

This stage represents the transition period between the stage of chemical peritonitis and that of bacterial peritonitis. Pathologically, it corresponds to the very early stage of bacterial peritonitis, and on the systemic side to the gradual transition from the stage of "peritoneal shock" to that of the toxæmia of bacterial infection.

The length of the stage of reaction shows considerable variation but rarely exceeds six hours. The most striking feature of this phase is the apparent improvement in the general condition of the patient—a feature which may lead to diagnostic difficulty. The patient experiences rather less spontaneous abdominal pain and feels generally better. He still lies completely motionless, however, and afraid to move. Instead of the blanched, ashen face of the precious stage, the lips and cheeks show signs of returning colour so that he actually looks better than he did some hours before. The temperature gradually returns to normal, the pulse shows very little change in frequency and the respirations remain slightly raised.

The abdominal physical signs show practically no alteration from those of the stage of chemical peritonitis. The retraction and immobility, the board-like rigidity,

the generalized tenderness and the alteration of the liver-dullness are all present. On percussion there may be an increase in the areas of dullness owing to the further exudation of peritoneal fluid.

The diagnostic difficulties in connection with this stage of the disease arise partly from the fact that the patient himself feels better and has rather less abdominal pain, partly from the fact that he looks better, and partly from the fact that his temperature and pulse show little variation from the normal. In these three respects his clinical condition shows considerable alteration from that of the preceding stage. On the other hand, the abdominal physical signs show scarcely any appreciable alteration. Few surgeons can have escaped being called to patients at this stage who have been put to bed without an exact diagnosis having been made. The stage of reaction is the stage of deception. The patient, on his journey from the preceding shock-like stage to the succeeding stage of bacterial peritonitis, passes for a few hours through a phase which in some respects brings him nearer to the normal. Mistakes will not occur if this phase of illusion is remembered, and adequate attention paid to the history of the case and especially to the abdominal physical signs.

Summary of Physical Signs in Various Stages.

	"Chemical peritonitis."	"Reaction."	"Bacterial peritonitis."	"Intestinal obstruction."
Facies	Ashen, anxious, cold and clammy	Looks better and of more normal colour	Cheeks flushed. Skin dry and hot. Tongue furred	Hippocratic, with sunken eyes, pinched nose and cold, clammy skin.
Temperature	Subnormal	Approximately normal	Moderate fever	Falling and becoming subnormal.
Pulse	Slightly increased, rarely above 90	As in previous stage or slightly increased	Rising	Increasing in frequency and becoming thin and thready.
Respiration	Thoracic in type, slightly increased in rate	Still thoracic and slightly increased in rate	Moderately increased in rate	Shallow and increasing in rate.
Vomiting	Nausea and vomiting usually absent	Absent	Vomiting commencing; gastric or duodenal in type	Progressive in amount and frequency and becoming fæculent.
Abdominal pain	Intense	Intense	Present and generalized but not as intense as in previous stages	Diminishing.
Appearance of abdomen	Retracted	Still retracted	No longer retracted. Commencing distension	Progressive distension.
Abdominal movements	Absent	Absent	Absent	Absent or paradoxical.
Rigidity	Board-like	Board-like	Generalized but not board-like	Diminishing and eventually disappearing.
Tenderness	Exquisite and generalized	Exquisite and generalized	Generalized	Gradually diminishing.
Liver-dullness	Diminished or absent	Diminished or absent	Diminished or absent	Diminished or absent.

FIG. 1.

THE STAGE OF BACTERIAL PERITONITIS.

The stage of bacterial peritonitis begins about 12 hours after perforation, and lasts for 24 hours or more until succeeded by the stage of paralytic intestinal obstruction. Bacteria multiply in the peritoneal cavity; the exudate is increased and becomes more frankly purulent; the peritoneal surfaces become hyperæmic and glued together by a variable amount of fibrinous exudate. At the same time the intestines become distended with gas and fluid and the intestinal movements diminish.

Although it is true that patients with peritonitis have severe abdominal pain, it is also true that patients with a perforated peptic ulcer get the most intense pain during the hours immediately following the perforation, and that with the passage of time this pain gradually diminishes so that the stage of paralytic obstruction may be almost painless. Abdominal pain is still present during the stage of peritonitis and is usually severe, but not as severe as during the hours immediately following perforation. The patient lies motionless in bed, but is no longer so terrified at the thought of even the slightest movement. Nausea and vomiting usually occur with the development of bacterial peritonitis. The facies at this stage are typically peritonitic. The eyes are bright, the cheeks flushed, the skin dry and the lips and tongue parched. A variable degree of fever is present, and the extent of this depends on the antecedent general condition of the patient. With patients in a good general condition the febrile reaction is marked, whilst in those who are wasted it may be comparatively slight. Throughout this stage the pulse gradually rises and the respirations remain increased.

Abdominal physical examination shows certain differences between this and the preceding stage. As the result of the accumulation of gas and fluid in the intestines the abdomen begins to get distended and, on inspection, the retraction which was formerly present has disappeared. Abdominal distension continues to increase until death. There is still no abdominal movement on respiration, and, on palpation, generalized rigidity is still present but is no longer board-like. It is the presence of generalized abdominal rigidity which limits for a time the distension. Tenderness is still present, but this also gradually tends to lessen. The diminution or obliteration of the liver-dullness still persists.

The Stage of Intestinal Obstruction.

The stage of intestinal obstruction begins about 36 hours after perforation and ends in the patient's death, usually at about 72 hours. This stage represents the paralytic stage of general peritonitis, and is clinically very similar to the terminal stages of intestinal obstruction from other causes. The intestinal paralysis is progressive, and the intestines become increasingly distended with gas and fluid. Pathologically there is a fall in the blood chlorides and a rise in the blood urea, following the loss of fluids from the body as the result of the profuse vomiting.

During this stage, spontaneous abdominal pain gradually diminishes. This is probably due to the dulling and exhaustion of nervous centres as the result of prolonged and intense peritoneal stimulation, and in part due to the gradual passing off of abdominal rigidity. It is during this stage that the patient begins to develop the characteristic Hippocratic facies, with the sunken eyes, the pinched nose and ears, and the ashen, clammy skin. This Hippocratic facies implies impending death, and the transition from the preceding peritonitic stage to the typical obstructive phase is a gradual one. Vomiting during this stage is frequent, profuse and usually without nausea. The patient turns his head to one side, and the intestinal contents simply pour forth without any obvious muscular effort or retching. As regards the actual material vomited, this is gastro-duodenal contents in the earlier stages, whilst in the later stages it becomes frankly fæculent. During the early part of the stage of intestinal obstruction a moderate degree of fever may still be present. On the whole, however, this gradually subsides, and during the terminal phase of his illness the patient has a subnormal temperature, which may be as low as 95°. At the same time the pulse shows a steady increase in frequency—an increase which began during the stage of reaction, was progressive throughout the stages of chemical and bacterial peritonitis, and in the obstructive stage may reach 140 to 160 per minute. The full bounding pulse of the peritonitic stage gradually gives way to the thin, thready, running pulse of the obstructive stage. At the same time there may be considerable increase in the frequency of the respirations, which become short and shallow partly on account of hypostatic changes in the lungs.

Abdominal examination shows progressive distension due to the collection of gas and fluid in the underlying bowel. There may be some movement of the abdomen wall with respiration, but this may be of a paradoxical

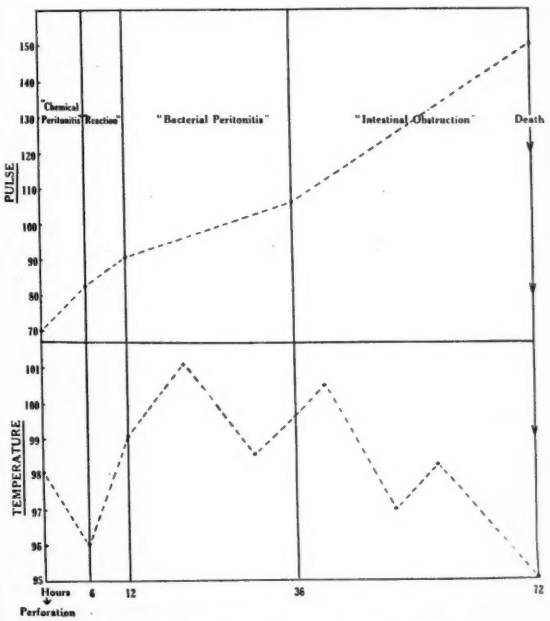

FIG. 2.—Diagram showing the general course of the temperature and the pulse in the various stages following perforation of a peptic ulcer into the general peritoneal cavity.

nature. On palpation, rigidity diminishes throughout this stage, and the generalized rigidity of peritonitis gradually disappears and is replaced by a flaccid abdominal wall. Similarly, tenderness also diminishes, and in the terminal phase of the disease may completely disappear. Dullness in the flanks and in the hypogastrium increases as the result of accumulation of fluid.

* * *

In taking the history of a patient with a suspected perforation of a peptic ulcer, inquiry will naturally be made as to the history of any antecedent ulceration. In many cases it is found that there has been an aggravation of abdominal symptoms and the associated

dyspepsia in the period immediately preceding perforation.

Although perforation usually takes place into the general peritoneal cavity, in a few instances it may be into the lesser sac. On the whole, however, ulcers of the posterior wall of the stomach show a marked tendency to fixation to the posterior abdominal wall, and in consequence, though they frequently give rise to gastric hæmorrhage, perforation is a rare complication. When the lesser sac of the peritoneum is involved in this way as the result of a perforation of a gastric ulcer, the abdominal symptoms may not necessarily be as acute or the physical signs be so generalized as when the general peritoneal cavity is involved. Some of the patients with this type of perforation may, after an initial acute abdominal phase, present the clinical picture of a subphrenic abscess. A detailed consideration of these lesser sac perforations is omitted from the present paper.

Prognosis in perforated ulceration depends primarily on the time at which operation is carried out. The longer the delay between perforation and operation, the higher is the mortality-rate. In cases coming under observation within a few hours of the development of acute symptoms, prognosis is good, but with each hour's delay after perforation the patient's chances of recovery steadily diminish. The stage of early bacterial peritonitis is still compatible with recovery, but the latter part of this stage and the stage of intestinal obstruction are uniformly fatal.

A realization of the pathological changes following the perforation of a peptic ulcer is essential to the understanding of the clinical aspects of the disease. The fact has been stressed that a patient with a perforated peptic ulcer does not long remain in an unchanged condition. With the object of simplifying the recognition of these changes the main features of the various stages have been stressed. The rapidity with which the patient passes from one stage to the next depends upon many factors, of which his general condition and resistance are probably the most important. On this account the actual duration of the various stages is of no great significance and may show very wide variations. In the stage of chemical peritonitis there should rarely be any doubt as to the diagnosis. It is in the stage of reaction which follows that a more serious diagnostic difficulty may be encountered. This stage of illusory and transient improvement has been discussed, but it is well to remember that during this stage the abdominal physical signs do not admit of any serious doubt as to the true state of affairs. The stage of bacterial peritonitis should not give rise to serious difficulty in diagnosis, but the condition may have to be differentiated from general peritonitis due to other causes. The final stage of the disease has to be differentiated from acute intestinal obstruction due to other causes, but the history, sequence and rapid progress of the case usually make the diagnosis clear.

Fig. 1 shows in diagrammatic form the essential features of the disease during the various stages, and Fig. 2 shows the general course of the temperature and the pulse.

REGINALD PAYNE.

STUDENTS' UNION.

CRICKET CLUB.

The following matches have been played since May 24th:

v. Romany C.C. *Lost* by 34 runs. Romany 234 (Rutherford 4 for 47, Harmer 5 for 97); St. Bartholomew's Hospital 200 (North 46, Harold 42, Harmer 40).

v. Leavesden Mental Hospital. *Lost* by 4 wkts. St. Bartholomew's Hospital 189 (North 49, Brown 33); Leavesden 206 for 7 wkts.

v. Croydon. *Lost* by 1 wkt. St. Bartholomew's Hospital 156 (Brown 42, Little 37); Croydon 164 (James 6 for 38).

v. Wanderers' C.C. *Drawn.* Wanderers 300 for 4 wkts. declared; St. Bartholomew's Hospital 181 for 3 wkts. (Johnstone 83, Heyland 72).

PAST *v.* PRESENT.

Played at Winchmore Hill on May 24th. The Present won by 5 wkts.

PAST.

Boney, b Mundy	5	Hunt, lbw, b Nicholson	24
Bourne, c Johnstone, b Mundy	6	Wilson, c Slowe, b Mundy	39
Wells Cole, c Wheeler, b Harmer	9	Maley, b Anderson	25
Gilbert, c Anderson, b Nicholson	49	Hay-Shunker, not out	13
Capper, b Nicholson	13	Extras	19
		Total (for 8 wkts., dec.)	202

Dransfield and Howell did not bat.

PRESENT.

Heyland, not out	86	Maidlow, b Wells-Cole	17
Johnstone, b Maley	25	Harmer, not out	28
Wheeler, b Maley	0	Extras	5
Brown, b Hay-Shunker	10		
North, b Hay-Shunker	41	Total (for 5 wkts.)	212

Mundy, Slowe, Nicholson and Anderson did not bat.

ST. BARTHOLOMEW'S HOSPITAL *v.* MIDDLESEX HOSPITAL.

2nd Round Inter-Hospitals Cup.

Played at Winchmore Hill on June 10th. *Won* by 181 runs.

Heyland, lbw, b Gethen	26	Harmer, c Abraham, b Gethen	44
Johnstone, b Skea	48	Hunt, not out	63
Wheeler, b Muir	8	Mundy, c Miller, b Abraham	11
Brown, b Thompson	0	Rutherford, not out	23
North, b Gethen	8	Extras	10
Maidlow, c Davies, b Thompson	32		
James, c Thompson, b Muir	7	Total (for 9 wkts., dec.)	280

MIDDLESEX HOSPITAL.

99 all out (James 4 for 41, Harmer 3 for 8).

LAWN TENNIS CLUB.

1ST VI.

1st Round Inter-Hospital Cup.

Played on Friday, May 22nd, at Winchmore Hill. St. Bartholomew's Hospital beat University College Hospital by 9 matches to *nil*.

Although U.C.H. did not win a match, we saw several close encounters. E. Corsi gave a most instructive exhibition of singles play when he beat J. F. Stokes 6—0, 6—2. W. K. Frewen just managed to beat A. L. Cochrane 6—3, 5—7, 8—6 after a long struggle. P. J. Hardie dropped the second set to G. H. Pearce before he got into his full stride, to win 6—2, 3—6, 6—1. H. R. Barnett beat J. R. Macleod 6—4, 5—7, 7—5, and G. L. Way beat H. M. Sinclair 6—2, 6—4. Thus, at the end of the singles Bart.'s were leading by 5 matches to *nil*, and all the doubles which were played also went the same way.

E. Corsi and W. K. Frewen beat J. F. Stokes and A. N. Cochrane 6—2, 7—5; beat J. H. Macleod and H. M. Sinclair 6—0, 6—1.

R. C. Witt and P. J. Hardie *v.* G. H. Pearce and H. A. Burt 7—9, 7—5, 9 all (unfinished).

G. L. Way and H. R. Marnett beat J. F. Stokes and A. L. Cochrane 7—5, 6—1; beat J. R. Macleod and H. M. Sinclair 6—2, 6—4.

PAST *v.* PRESENT.

On Saturday, May 23rd, the Past were defeated by the Present by 3 matches to 6. In a very enjoyable match E. Corsi and W. K. Frewen beat J. Beattie and C. H. Evans 6—0, 6—1; beat J. H. Hunt and H. E. Archer 6—1, 6—3; beat O. S. Tubbs and R. Marnett 6—1, 6—1.

R. C. Witt and P. J. Hardie beat J. Beattie and C. H. Evans 6—2 6—1; beat J. H. Hunt and H. E. Archer 3—6, 9—7, 6—2; lost to O. S. Tubbs and R. Marnett 2—6, 2—6.

H. R. Marnett and M. H. Desmarais beat J. Beattie and C. H. Evans 9—7, 5—7, 6—3; lost to J. H. Hunt and H. E. Archer 6—0, 5—7, 4—6; lost to O. S. Tubbs and R. Marnett 5—7, 0—6.

SWIMMING CLUB.

Inter-Hospital Water-Polo League.

Results:

v. St. Thomas's Hospital; away. Won, 11—3.
v. St. Mary's Hospital; home. Won, 8—3.
v. Dental and Charing Cross Hospital; away. Won 7—0.
v. University College Hospital; home. w.o.
v. Guy's Hospital; away. Lost, 3—5.
v. London Hospital; home. Won, 13—0.

The results speak for themselves. All of the games were won easily with the help of Sutton, Newbold, Norton and McKane, who were the principal scorers. The unfortunate lapse against Guy's was due to the weak team we fielded, owing to the demands of county matches on some and lack of enthusiasm among other members of the Club.

Other Fixtures.

Results:	Polo.	Swimming.
v. Lensbury; away	Won, 3—2	Won.
v. Old Millhillians; away	Won, 5—1	Lost.
v. St. Paul's School; away	Won, 6—3	Lost.
v. Epsom College; away	Scratched.	
v. Cambridge Tadpoles; away	Lost, 2—6	Won.
v. St. Mary's Hospital; away	Lost, 3—6	Lost.

Owing to the late start of the season occasioned by making new bath arrangements only five matches have been played, three being scratched. The swimming has been fair. We beat the Tadpoles 17—14; Newbold and Pratt came first and second in the 100 yards, Singer second in the 50 yards, and Evans won the diving.

Lack of training has lost us the swimming on several occasions. With the better facilities offered to members now at St. Mary's, where we have the bath once a week entirely to ourselves, more keenness would obviate these failures.

COLLEGE APPEAL FUND.

SUBSCRIPTIONS TO DATE.

	£ s. d.	*	†
Staff	13,404 1 4	(78)	
Demonstrators	1,774 17 0	(72)	
Students	1,224 13 1	(324)	
Old Bart.'s men:			
‡Bedfordshire	45 18 6	(9)	(26)
Berkshire	123 3 0	(16)	(37)
‡Buckinghamshire	82 4 0	(15)	(29)
‡Cambridgeshire	194 6 0	(18)	(42)
‡Cheshire	6 16 6	(3)	(26)
‡Cornwall	22 12 0	(8)	(36)
Cumberland	5 0 0	(1)	(6)
Derbyshire	19 14 0	(4)	(17)
‡Devonshire	575 1 0	(54)	(98)
‡Dorset	77 11 6	(14)	(30)
‡Durham	17 7 0	(4)	(11)
Essex	267 3 6	(23)	(69)
‡Gloucestershire	257 5 6	(29)	(52)
Hampshire	1,517 4 6	(59)	(134)
‡Herefordshire	17 12 0	(4)	(10)
Hertfordshire	91 18 0	(19)	(73)
Huntingdonshire	5 5 0	(1)	(1)
Isle of Wight	191 13 0	(13)	(25)
‡Kent	588 5 0	(72)	(146)
‡Lancashire	127 14 6	(16)	(82)
Leicestershire	142 0 0	(8)	(28)
‡Lincolnshire	61 9 0	(18)	(27)
‡Middlesex	497 14 0	(34)	(63)
‡Norfolk	178 0 6	(21)	(60)
‡Northamptonshire	59 14 6	(6)	(17)
‡Northumberland	101 1 0	(2)	(11)
‡Nottinghamshire	24 3 0	(5)	(28)
‡Oxfordshire	231 15 0	(22)	(26)
Rutland	1 1 0	(1)	(2)
Shropshire	38 1 0	(10)	(22)
‡Somersetshire	2,837 6 4	(28)	(43)
Staffordshire	194 18 0	(6)	(37)
‡Suffolk	331 0 6	(26)	(46)
Surrey	523 18 6	(62)	(180)
Sussex	752 4 6	(63)	(174)
‡Warwickshire	214 19 0	(24)	(64)
Westmorland	2 10 0	(1)	(5)
‡Wiltshire	1011 12 0	(13)	(26)
‡Worcestershire	161 1 6	(25)	(25)
‡Yorkshire	350 3 6	(29)	(101)
Wales	69 12 0	(20)	(150)
London	6,851 5 2	(228)	(971)
Channel Islands	20 0 0	(2)	(9)
Scotland	15 5 0	(5)	
Abroad	119 1 0	(13)	
South Africa	376 15 6	(20)	
Canada	114 3 6	(8)	
East Africa	87 12 0	(10)	
West Africa	146 10 0	(5)	
India	207 12 0	(13)	
Ireland	25 4 0	(4)	
North Africa	1 0 0	(1)	
North Borneo	10 10 0	(1)	
Australia	122 2 0	(6)	
China	52 8 4	(9)	
Siam	10 0 0	(1)	
France	50 0 0	(1)	
British West Indies	65 8 0	(7)	
Straits Settlements	7 1 0	(3)	
New Zealand	6 1 0	(3)	
Services	654 14 6	(49)	
Others	71,377 9 7	(568)	
Lord Mayor's Appeal	17,990 16 0		
Funds of College	8,000 0 0		
Value of Building	20,000 0 0		
Loan	20,000 0 0		
Stock Sold	4,061 0 0		
	£178,793 9 4		

* Number of Bart.'s men subscribing. † Number of Bart.'s men in County. ‡ Counties with Secretaries.

TIMES OF ATTENDANCE IN X-RAY DEPARTMENT: A CORRECTED LIST.

	Monday.	Tuesday.	Wednesday.	Thursday.	Friday.	Saturday.
Diagnostic . .	Dr. Loughborough at 9.30 a.m. Dr. Sparks at 1.30 p.m.	Dr. Finzi at 9.30 a.m. Dr. Stone at 1.30 p.m.	Dr. Stone at 9.30 a.m. —	Dr. Loughborough at 9.30 a.m. Dr. Stone at 1.30 p.m.	Dr. Sparks at 9.30 a.m. Dr. Loughborough at 1.30 p.m.	Dr. Sparks at 9.30 a.m. —
Therapeutic .	Dr. Finzi at 1.30 p.m. Dr. Levitt at 2.30 p.m.	Dr. Levitt at 1.30 p.m. —	Dr. Levitt at 9.30 a.m. —	Dr. Levitt at 1.30 p.m. —	Dr. Levitt at 9.30 a.m. Dr. Finzi at 1.30 p.m.	— —

CORRESPONDENCE.

JOHN MELLY.

To the Editor, 'St. Bartholomew's Hospital Journal'.

SIR,—It appears to me fitting that the work and self-sacrifice of John Melly should be commemorated in this Hospital by some form of memorial, and I should like to suggest that the matter should be considered by the authorities. I am certain that many of his friends, both medical and others, would be glad to contribute to such a memorial. Should this already have been taken in hand I must apologize for troubling you.

Yours, etc.,

O. R. TISDALL.

Westfield, West Hill,
Harrow-on-the-Hill;
June 4th, 1936.

THE MASSAGE DEPARTMENT.

To the Editor, 'St. Bartholomew's Hospital Journal'.

SIR,—It would appear that some of your readers have interpreted my article on the Massage Department as a history of that Department. This was far from being my intention when I wrote the article. A history of the Department would have to include its shifting of position from the old Surgery, where it started, to the new Out-Patient Quarters (now the X-Ray Department), and thence after long-drawn-out negotiations to its present locale in the Medical Out-Patient Department. This site, intended to be temporary, has remained permanent for more years than I care to remember, and is likely to last out my time. And I should also, in an historical summary, have mentioned more of the staff of the Department. From 1908 onwards we have had a sister-in-charge of the Department, first Miss Vergette, who was responsible for its organization, and who (with two or three nurse-pupils) had also to treat all the in-patients; later Miss McCulloch Smith, who now has charge of the fully trained staff. Lieut. Tham also had male assistants and successors, and at one time we had blind pupils from St. Dunstan's attending. But as I have already said, these historical details were not really within the scope of my article, which was intended to be a guide to the present-day student, and to help him to understand the position and training of the massage profession.

Yours, etc.,

R. C. ELMSLIE.

1A, Portland Place, W. 1;
May 26th, 1936.

THE TEACHING OF THERAPEUTICS.

To the Editor, 'St. Bartholomew's Hospital Journal'.

SIR,—A certain gentleman has put in a plea for more space in our clinical lectures to be devoted to treatment. Although a student myself I cannot support this plea.

May I point out to him that the basic function of this Hospital, from the teaching point of view, is to teach diagnosis rather than treatment? As we all know, or will very soon find out, rational treatment is quite impossible without accurate diagnosis. Therefore it neither surprises me nor worries me that our lecturers spend thirty-five of their allotted forty minutes on the latter subject.

When we reach the stage of medical practice we can "look up" the question of treatment in a great many of our cases; whereas it is a very different matter to obtain a diagnosis from any book.

It is essential that we should spend by far the greater part of our time here in developing "clinical sense" and learning the art of diagnosis. It is in the very best interests of our patients, even if we come to regard treatment as a mere after-thought.

I remain, Sir,

Yours faithfully,

C. M. DICKINS.

REVIEWS.

SYMPTOMS AND SIGNS IN CLINICAL MEDICINE: AN INTRODUCTION TO MEDICAL DIAGNOSIS. By E. NOBLE CHAMBERLAIN, M.D., M.Sc., M.R.C.P. With a chapter on "The Examination of Sick Children", by NORMAN B. CAPON, M.D., F.R.C.P. (Bristol: John Wright & Sons, Ltd., 1936.) Pp. 424. 282 illustrations, of which 17 are in colour. Price 25s.

When the student begins his study of clinical medicine, he is frequently beset with the difficulties of what questions he should ask the patient and what signs he should look for in the elucidation of the diagnosis. This book amply supplies his needs. The first chapter describes the method of obtaining a history from the patient, and contains a valuable table setting out the line of investigation the medical man should adopt in endeavouring to determine the cause of the patient's complaints. Subsequent chapters deal in more detail with the symptoms and signs appertaining to systemic disease. The tedium, however, of memorizing symptoms and signs is relieved at the end of each chapter by a brief description of the commoner and more important diseases of that particular system, whereby the student may learn how his findings may be pieced together in arriving at a diagnosis. Mention is also made of the special laboratory and instrumental investigations which may be necessary in the complete examination of the case, and those simpler ones which may be carried out by the general practitioner. The value of the book is greatly enhanced by the excellent illustrations and radiograms, which show better than words can describe the methods of examination and the diseased states which may be encountered.

A MANUAL OF PRACTICAL OBSTETRICS. By O'DONEL BROWNE, M.B., B.Ch., B.A.O., F.R.C.P.I., L.M., M.C.O.G. (Bristol: John Wright & Sons, Ltd.) Pp. 363. 10 plates, 236 illustrations. Price 20s.

The first thing which strikes one about this volume is that it is small. It has been kept so purposely, for it is intended to be of practical assistance to the student and the practitioner. The book is well printed and clearly illustrated, and the majority of the matter contained is tabulated so that it can easily be used for reference. The diet charts and other lists will be useful to the practitioner. There are opinions in the book which are at variance with the London teaching, notably the advocacy of induction of labour at the thirty-sixth week in contracted pelvis in primiparæ, the application of the forceps to the breech, and the encouragement given to packing the uterus in cases of post-partum hæmorrhage. There are other smaller points which will probably mean that the book will be more widely used in Dublin than in London.

1. MINOR SURGERY AND THE TREATMENT OF FRACTURES. By GWYNNE WILLIAMS, M.S., F.R.C.S. Twenty-first edition. (Messrs. J. & A. Churchill, 1936.) Price 10s. 6d.

2. ELEMENTARY SURGICAL HANDICRAFT. By J. RENFREW WHITE, Ch.M., F.R.C.S., F.A.C.S. (Messrs. J. & A. Churchill, 1936.) Price 8s. 6d.

The new edition of Mr. Glynne Williams's excellent little book appears under a slightly different title, and the inclusion of the section on the treatment of fractures will be of great value to the house surgeon. All common fractures are briefly but adequately dealt with, and the modern methods of plaster technique and skeletal traction are a feature. One is, however, a little disappointed that no mention is made of the three-handkerchief method of treating fracture of the clavicle, although Sayre's method is described at some length. The chapter on anæsthetics has been very ably written by Dr. H. N. Webber, and he contributes much useful information on basal narcotics and methods of local anæsthesia. Interest is added by a short note on the original author and his successors, together with a portrait.

In comparison with this popular book, Mr. Renfrew White has not been so successful in his unenviable task of trying to impart a knowledge of bandaging and other minor surgical procedures, due partly to the poorness and inadequacy of the illustrations, and partly to the rather bewildering variety of methods, many of which are unlikely ever to be used. The mechanical tourniquets described in the chapter on hæmorrhage, for instance, hardly find a place in modern surgical practice.

PRACTICAL POINTS IN ANÆSTHESIA: A CLINICAL HANDBOOK FOR STUDENTS AND GENERAL PRACTITIONERS. By H. K. ASHWORTH, M.B., L.R.C.P.(Lond.), D.A.(R.C.P.&S.Eng.). WITH A CHAPTER ON LOCAL ANÆSTHESIA by H. T. SIMMONDS, B.Sc., M.B., Ch.B. (Vict.), F.R.C.S. (J. & A. Churchill, 1936.) Price 7s. 6d.

This little book is a reprint of a series of articles published in the *Manchester University Medical School Gazette*, and is intended for the beginner.

With this object in view the book is intentionally elementary, theoretical considerations are minimized, and practical points emphasized by paragraph headings in black type.

The early chapters are the best part of the book, and those on pre-operative considerations, induction, maintenance and complications of anæsthesia are clearly written, and contain many helpful hints on the difficulties that may be encountered and how they may be overcome.

The chapter on nitrous oxide is definitely inadequate for the needs of the modern student. The account of nitrous oxide-oxygen anæsthesia is sketchy, while there is no mention whatsoever of the use of nitrous oxide (without oxygen) for minor operations or for dental work. The author apparently subjects all his dental cases to a C.E. mixture. Surely one of the most frequent anæsthetics the general practitioner is called upon to give is the straightforward dental gas?

The chapter on Local Anæsthesia is good, but why include in a book of this type such detail as an account of splanchnic block anæsthesia?

This is nevertheless a book which the student can read with advantage to himself and increased safety to his patients, and could well be perused in a corner of the anæsthetic room while waiting his turn "at the chin".

"HERO-DUST." By JAMES KEMBLE, Ch.M., F.R.C.S. (London: Methuen, 1936.) Price 6s. net.

I must confess some little disappointment with Mr. James Kemble's new book, *Hero-Dust*, in which he carries on the same line of thought which made his *Idols and Invalids* such an amusing and authentic piece of historical indiscretion. For although he ostensibly sets out to deal with his diverse characters—Mary, Queen of Scots, Epicurus, Catherine the Great, John Milton, Beau Brummell, and Omar Khayyám—in the same way as before, laying the responsibilities for their historical acts upon their disordered physiologies, the potted history is rather too generous, and the pathology, in several cases, all too thin.

Beyond recording the fact that Epicurus had a vesical stone and Beau Brummell died of a stroke, he makes no attempt to link their destinies with their diseases. And it is difficult to see why Omar, who had a perfectly clean bill of health, and died a centenarian, is there at all, unless it is for an exposition of the *Rubáiyát*, which has been done before and done better.

Nevertheless the book is well written, and even if the author's humour becomes at times a shade facetious, it is often amusing—two qualities not to be sneezed at in these days of literary incompetence and incontinence.

A MANUAL OF EMERGENCIES—MEDICAL, SURGICAL AND OBSTETRIC: THEIR PATHOLOGY, DIAGNOSIS AND TREATMENT. By J. SNOWMAN, M.D., M.R.C.P. Third edition. (John Bale, Sons & Danielsson, 1936.) Price 10s. 6d.

The author's most laudable object may be gathered from the title of this book: a book of pocket size, intended to contain all the emergencies with which the general practitioner is likely to be faced. Important differential diagnoses are given in tabular form and prescriptions are both numerous and detailed. Heavy type is well used to catch the hurried eye. A suggestion for further improvement would be the inclusion in the index of important symptoms as an aid to diagnosis. This book is certainly unique in its scope and should continue to be of great service both to "housemen" and general practitioners. The third edition contains many recent advances in treatment.

ELEMENTARY ZOOLOGY FOR MEDICAL STUDENTS. By L. A. BORRODAILE, Sc.D. Third edition. (Oxford Medical Publications, 1935.) Pp. 429. Price 10s. 6d.

This new edition of a text-book with so well-established a reputation will receive a welcome in many quarters. The careful revision which it has received has improved the volume considerably, and has brought its contents more completely into line with the "agreed syllabus" in biology of the Universities of Oxford, Cambridge and London. In this respect it forms a zoological counterpart to the *Elements of Plant Biology* by Tansley and James, which was reviewed in our last issue. We can assure our readers that the work is eminently suitable for those preparing for the First Medical Examination.

AN INTRODUCTION TO HYGIENE. By W. ROBERTSON, M.D., D.P.H., F.R.C.P.(Ed.). Second edition. (E. & S. Livingstone, 1936.) Pp. xv + 314. Price 8s. 6d.

In his introduction Dr. Robertson says "Books act as guides", and certainly this book should be sufficient to guide a student through the university qualifying examinations. Out of his wide experience, both as medical officer of health and lecturer and examiner in the subject, has come a concisely written book dealing in an easily understood manner with the various aspects of preventive medicine. There is also some very good advice in the introduction to those about to embark on a medical career. Apart from minor faults, such as an annoying habit of splitting infinitives and a reference to Hunterian sore as soft, this is a book to recommend for any medical man's library. The book is well produced, the print is large and clear and the paragraphs are well emphasized, so that it is easy to see the subject-matter at a glance.

THE CHEMICAL CONTROL OF CONCEPTION. By JOHN R. BAKER, M.A., D.Phil. (Chapman & Hall, 1936.) Pp. x + 173. Price 15s.

In this small book Mr. Baker has presented the results of many years' well-thought-out and carefully controlled experiments. First he describes how a physiological solution in which the tests could be performed was obtained; and how cavy sperms, which he used in most of the tests, were found to respond to widely varying types of stimuli in an almost identical manner to human sperms. Many substances, mostly of a complicated formula developed from already accepted spermicides, were tested and the criteria of their potency described in detail. Several of the common suppositories and pessaries sold as contraceptives were also tested, with rather depressing results. A chapter is added on the pathology of contraception by H. M. Carleton, and tables are appended comparing the results of the tests. Altogether a very excellent book, although rather for the biochemist or specialist than for the average doctor.

EXAMINATIONS, ETC.

University of Cambridge.

The following Degrees have been conferred :

M.D.—Bourne, W. A., Burrows, H. J.
M.B., B.Chir.—Gordon, C. J., Martin Jones, J. D.
M.B.—Patterson, J. H., Tracey, J. B.
B.Chir.—Black, K. O., Briggs, W. A., Darmady, E. M.

University of London.

Third (M.B., B.S.) Examination for Medical Degrees, May, 1936.

Honours.—*Clarke, E. P.

* Distinguished in Surgery.

Pass.—Anderson, C., Beach, H. L. W., Hartsilver, J., Kanaar, A. C. Martin, J. R. M., Ogilvie, J. D., Oliver, W. A., Roberts, J. C., Sheehan, D. J., Taylor, G. R.

Supplementary Pass List.

Group I—Fearnley, J. D. O.

Group II.—Barnard, E. J. W., Brentnall, G. C., Brown, K. P., Cochrane, J. W. C., Gibson, R. E., Grundy, T. N., Leask, L. R., McGladdery, J. P., Roberts, J. L. D., Rotter, K. G., Samuel, D. M.

University of Witwatersrand.

The following Diploma has been conferred :

D.P.H.—Clark, B. M.

CHANGES OF ADDRESS.

CAMMIDGE, P. J., 85, North Gate, Regents Park, N.W. 8. (Tel. Primrose 6211.)
DE CAUX, F. P., 30, Green Street, Mayfair, W. 1. (Tel. Mayfair 7177.)
ECCLES, W. MCADAM, 104, Bryanston Court, Upper George Street, W. 1. (Tel. Paddington 7499—unaltered.)
HARRIS, CHARLES F., The Warden's House, St. Bartholomew's Hospital Medical College, Charterhouse Square, E.C. 1. (Tel. Clerkenwell 4616.) [31, Weymouth Street, W. 1. (Tel. Langham 1723)—unchanged.]
MASTERMAN, E. W. G., English Mission Hospital, es Salt, Trans-Jordan.
PRATT, ELDON, 3, Dorchester Mansions, Manor Road, Bournemouth.
SHORE, T. W., "Dunrobin", Vapron Road, Plymouth. (Tel. Plymouth 5170.)
TAYLOR, G. C., 3, Kingsdale Road, Berkhamsted, Herts.

APPOINTMENTS.

HUNT, W., M.R.C.S., L.R.C.P., appointed Certifying Factory Surgeon for the Carlton District, Nottingham.
SHARP, B. BUCKLEY, M.D., M.R.C.P., appointed Honorary Consulting Physician to Acton Hospital.

BIRTHS.

ARCHDALE-SMITH.—On May 21st, 1936, at 31, Brunswick Road, Hove, to Peggy (*née* Lacey), wife of David Archdale-Smith, M.B. —a son.
DAY.—On May 22nd, 1936, at 17, Chapel Field East, Norwich, to Dr. and Mrs. George Day—a son.
GARSON.—On June 3rd, 1936, at 66, Rodney Street, Liverpool, to Nan, wife of Philip Garson—a daughter.
HOBBS.—On June 12th, 1936, at Old Court, Ealing, to Agnes, wife of Dr. A. N. Hobbs, 30, Woodville Road, W. 5—a son.
HOSFORD.—On May 27th, 1936, at Cairnton, Stormont Road, Highgate, to Nora (*née* Randall), wife of R. W. P. Hosford, F.R.C.S.—a daughter.
JORY.—On June 3rd, 1936, at 2, Broadlands Road, Highgate, to Daphne, wife of Norman Jory, F.R.C.S.—a daughter.
PERROTT.—On June 5th, 1936, at Suffolk House, Stanmore, to Louie, wife of Dr. G. F. Donaldson Perrott—a son (John Anthony).
RADCLIFFE.—On May 24th, 1936, at 102, Rockingham Road, Kettering, to Phyll, wife of Frank Radcliffe, M.B., F.R.C.S.—a son.
SAWLE THOMAS.—On June 9th, 1936, at 20, Devonshire Place, W. 1, to Maryon, wife of J. Sawle Thomas, M.R.C.S.Eng.—a daughter.
TANNER.—On May 30th, 1936, at the Melvin Nursing Home, Newton Abbot, to Nancy (*née* Thynne), wife of Dr. Guy Montague Tanner —a son.
WILKIN.—On June 11th, 1936, at 50, London Road, Gloucester, to Margaret Annette (*née* Graham), wife of W. J. Wilkin, F.R.C.S. —a daughter.

MARRIAGES.

FURBER—CRAWFURD.—On May 30th, 1936, at St. Peter's, Earley, Reading, Lionel Brian Furber, son of Dr. and Mrs. Furber, of Twyford, Berks, to Gwendolen Mary Payne Crawfurd, daughter of Lt.-Colonel and Mrs. Crawfurd, Merrows, Westcote Road, Reading.
HUDSON—ADLINGTON.—On June 9th, 1936, in London, Bernard Hudson, M.D., M.R.C.P., of Davos, Switzerland, to Laura Almon, younger daughter of Mr. and Mrs. Adlington, of Bath.
LOCKETT—BRADING.—On June 6th, 1936, at Emmanuel Church, Croydon, Dr. John Morton Lockett, M.R.C.S., L.R.C.P., eldest son of Mr. and Mrs. A. W. Lockett, of Croydon, to Barbara Cicely, youngest daughter of Mr. and Mrs. L. H. Brading, of Sutton, Surrey.
MAXWELL—EVANS.—On June 4th, 1936, at St. Bartholomew-the-Great, Smithfield, quietly, by the Rt. Rev. Bishop Paget, James Maxwell, M.D., F.R.C.P., to Elizabeth Nan Evans.
PATTERSON—YEAMES.—On June 10th, 1936, at Helen's Bay Church, by the Rev. L. Martin, B.A., Dr. Joseph Henry Patterson, elder son of the Hon. Mr. Justice D. C. Patterson and Mrs. Patterson, Calcutta, to Hélène Sobolevski, second daughter of Mr. and Mrs. Yeames, Old Mill House, Helen's Bay, co. Down.
ROBERTS—JOHNSON.—On June 10th, 1936, at St. Mary's, Dunmow, by the Rev. E. Noel Mellish, V.C., M.C., Vicar, Arthur Michael, elder son of the late Arthur H. Roberts, West Malling, Kent, to Freda Mary, youngest daughter of the late Walter Johnson, of Terrington St. John's, Norfolk.
SLOT—LEWIS.—On May 29th, 1936, Gerald Slot, M.D., of 117A, Harley Street, W. 1, to Mary Lewis, of 2, Wellington House, Regent's Park, N.W. 8, daughter of the late Mr. and Mrs. J. A. Munton, of Idle, Yorkshire.

DEATHS.

COPE.—On June 5th, 1936, Albert Ernest Cope, M.D., B.S., D.P.H., late of 66, Belgrave Road, Westminster, aged 68.
DAVIES.—On June 11th, 1936, suddenly, in London, Jenner Conway Davies, M.B., B.Ch., M.R.C.P., youngest son of the late Dr. G. A. Davies, of Newport, Mon., and Mrs. Davies.
GILMOUR.—On June 10th, 1936, at The Cedar House, Woodbridge, Percy Graham Gilmour, M.R.C.S., L.R.C.P.
HURTLEY.—On June 2nd, in St. Bartholomew's Hospital, William Holdsworth Hurtley, of 28, Chestnut Road, S.E. 27, aged 70.
NELSON.—On June 24th, 1936, at St. Bartholomew's Hospital, Henry Philbrick Nelson, M.D., F.R.C.S., beloved husband of Kathleen Nelson, aged 34.
WORTH.—On June 24th, 1936, at Bar Helford Passage, Falmouth, Claud Worth, F.R.C.S. (late 34, Harley Street).

NOTICE.

All Communications, Articles, Letters, Notices, or Books for review, should be forwarded, accompanied by the name of the sender, to the Editor, ST. BARTHOLOMEW'S HOSPITAL JOURNAL, *St. Bartholomew's Hospital, E.C. 1.*

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